A STUDY PROPOSAL ON SHORT TERM OUTCOME AND PROGNOSIS OF PRIMARY & DELAYED FULGURATION IN POSTERIOR URETHRAL VALVE

Dissertation Submitted to

THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations

For the award of the degree of

M.Ch. Branch V

PEDIATRIC SURGERY

2008 – 2011

MADRAS MEDICAL COLLEGE & RI

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The Institutional Review Board [Ethical committee] of Institute of Child Health and Hospital for Children, Chennai-08, was held on 30.01.2010 at 10.00AM at the Deputy Superintendents chamber.

Members Present: Dr.R.Kulandai Kasthuri
Chair Person.

Members:
1. Dr.K.Gita
2. Dr.P.Jeyachandran
3. Dr.D.Vijaya Sekaran
4. Prof.Girija Shyam Sundar
5. Mrs.Muthu Lakshmi, (Advocate)
6. Dr.P.Ramachandran
7. Mrs.Shubha Kumar

Member Secretary: Dr.Luke Ravi Chellaiah

Title: " A Study of Comparing short Term outcome of Primary Fulgration Vs Diversion and Delayed Fulgration in Posterior Urethral Valve ".

The Institutional Review Board was satisfied with the revised format submitted by you. Hence the Institutional Review Board is pleased to approve the study.

Director and Superintendent.

To,
Dr.P.Jai Durairaj,
M.ch Post Graduate,
ICH & HC,
Chennai-08.

CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON SHORT TERM OUTCOME AND PROGNOSIS OF PRIMARY & DELAYED FULGURATION IN POSTERIOR URETHRAL VALVE"** is a bonafide work done by Dr. P.JAI DURAI RAJ under my guidance and supervision during the period between 2008-2011 towards the partial fulfillment of requirement for the award of M.Ch Branch V (Paediatric Surgery) degree examination held in August 2011 by The Tamilnadu Dr.M.G.R. Medical University, Chennai.

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DECLARATION

I solemnly declare that the dissertation entitled **"A STUDY ON SHORT TERM OUTCOME AND PROGNOSIS OF PRIMARY & DELAYED FULGURATION IN POSTERIOR URETHRAL VALVE"** is the original work done by me at the Institute of Child Health and Hospital for Children, Egmore, during the M.Ch. course (2008-2011), under the guidance and supervision of Prof.S.V.Senthilnathan M.S., M.Ch. Professor and H.O.D. of Paediatric Surgery. The dissertation is submitted to **THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY** towards the partial fulfillment of requirement for the award of **M.Ch. (BRANCH – V) in PAEDIATRIC SURGERY.**

Place: Chennai. Dr. P.JAI DURAIRAJ

Date: 30/5/2011

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CONTENTS

A STUDY ON SHORT TERM OUTCOME AND PROGNOSIS OF PRIMARY & DELAYED FULGURATION IN POSTERIOR URETHRAL VALVE

INTRODUCTION

PUV is the most common cause of Lower Urinary Tract obstruction in male neonates. The incidence is I in 4000- I in 7500 births PUV occur exclusively in males. This disease has a broad spectrum of presentation. They may present at any age during childhood & may vary from ascites in the neonate to renal failure in infant and only minor voiding dysfunction in an older child. Urinary tract infection is common at all ages.

Oligohydramninos & hydronephrosis detected by antenatal ultrasound may be associated renal dysplasia which may compromise renal function despite relief of obstruction.

The diagnosis is made by ultrasound & micturating cysto-urethrography. To know the renal status and to evaluate the Urinary tract infection. Sr. Creatinine, blood urea & urine c/s will give a clue.

Surgical care of the patient with PUV varies according to age, bladder status & renal status ideal treatment involves trans urethral ablation using electro cautery of the PUV(Fulguration). When Urethral size precludes valve ablation,a vesical (i.e. vesicostomy) or supra-vesical

(ureterostomy)diversion can be created to provide upper tract drainage. Bilateral Ureterostomy can also be placed for urinary drainage in certain condition.

PUV present with wide spectrum of renal & bladder pathology including damage to renal parenchyma as well as to smooth muscle function of urinary bladder. These changes may persist despite successful management of the primary obstructing PUV leading to bladder dysfunction & renal insufficiency, which is the primary cause of morbidity & mortality in these children.

Much work has been done to identify the factors that predict the final outcome in these cases so as to prognosticate the end result and implicated the correct treatment protocol. This study reviews retrospectively a series of 35 children with posterior urethral valves who completed all stages of treatment presented at our institute over the last 3-5 years with an aim to identify the prognostic factors prospectively.

AIM AND OBJECTIVES OF STUDY

1. TO ASSESS THE IMPACT OF PRIMARY IMPACTION ON SHORT TERM OUTCOME.

2. TO ASSESS THE OUTCOME OF DIVERSION AND DELAYED FULGURATION.

3. TO TEST THE EFFICIENCY OF THE TREATMENT.

4. TO IDENTIFY THE PROGNOSTIC FACTORS.

INCLUSION CRITERIA

1. POSTERIOR URETHRAL VALVE DISEASE DIAGNOSED AND TREATED WITH PRIMARY FULGURATION IN CHILDREN/INFANT & NEWBORN.

2. POSTERIOR URETHRAL VALVE DISEASE DIAGNOSED AND TREATED WITH DIVERSION ,DELAYED FULSURATION AND DIVERSION CLOSURE IN CHILDREN/INFANT & NEWBORN

EXCLUSION CRITERIA

1. POSTERIOR URETHRAL VALVE PRESENTED WITH RENAL INSUFFICIENCY

2. PRUNE BELLY SYNDROME WITH POSTERIOR URETHRAL VALVE.

3. POST PROCEDURAL COMPLICATIONS LIKE STRICTURE, DIVERSION WERE NOT TAKEN IN THIS STUDY.

PATIENTS AND METHODS

35 children with posterior urethral valves presented during the period of 2008 – 2010. The average duration of follow-up was 3 years.

A routine haemogram, routine urine analysis and culture examination, serum creatinine and electrolytes, ultrasound examination and micturating cystourethrogram (MCUG) constituted the baseline investigations.All the patients were put on a per – urethral cathethar drainage at admission, which was continued till the serum creatinine level stabilized, with 2 consecutive levels showing no further drop. This was followed by primary valve fulguration and continuous chemoprophylaxis.

At follow – up, weight and height estimation of the child, urine routine and culture analysis, serum creatinine estimation were done to assess the progress of the child for every 3 months in 1st year, and every 6 months for 3yrs.

Ultrasound to assess the size of kidney and corticomedullary differentiation, size of ureter,

bladder wall thickening, post void residual urine volume for every 3 months in 1 year and every 6 months for 3 yrs. MCUG was done every 6 monthly follow-up.

Indication for a urodynamic evaluation included persistent upper tract dilatation or rising serum creatinine in spite of an adequate bladder drainage and persistent voiding dysfunction after an adequate valve fulguration.

Indication for DMSA were persistent high sr. cretinine level and parenchymal changes in ultrasonography

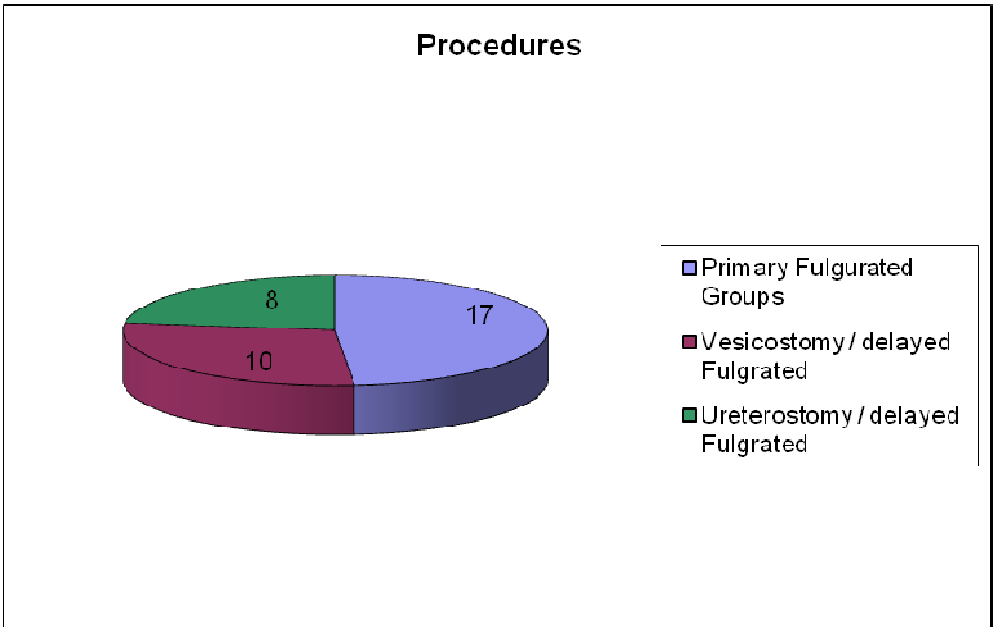
Factors that could play a role in the final outcome were studied. These included age at presentation and intervention (less than or more than 2 years), recurrent urosepsis (3 or more/year), persistence of vesico – ureteric reflux, renal parenchymal damage as seen on ultrasound, vesical dysfunction and the nadir serum creatinine level.

The no of children who undergone the procedures for confifmed posterior urethral valve in our series:

Fulgurated groups – 17 children

Vesicotomy diversion – 10 children

Ureterostomy diversion – 8 children in which B/L for 2 cases and U/L for cases.



PRIMARY FULGRATION GROUP

DIVERSION GROUP:

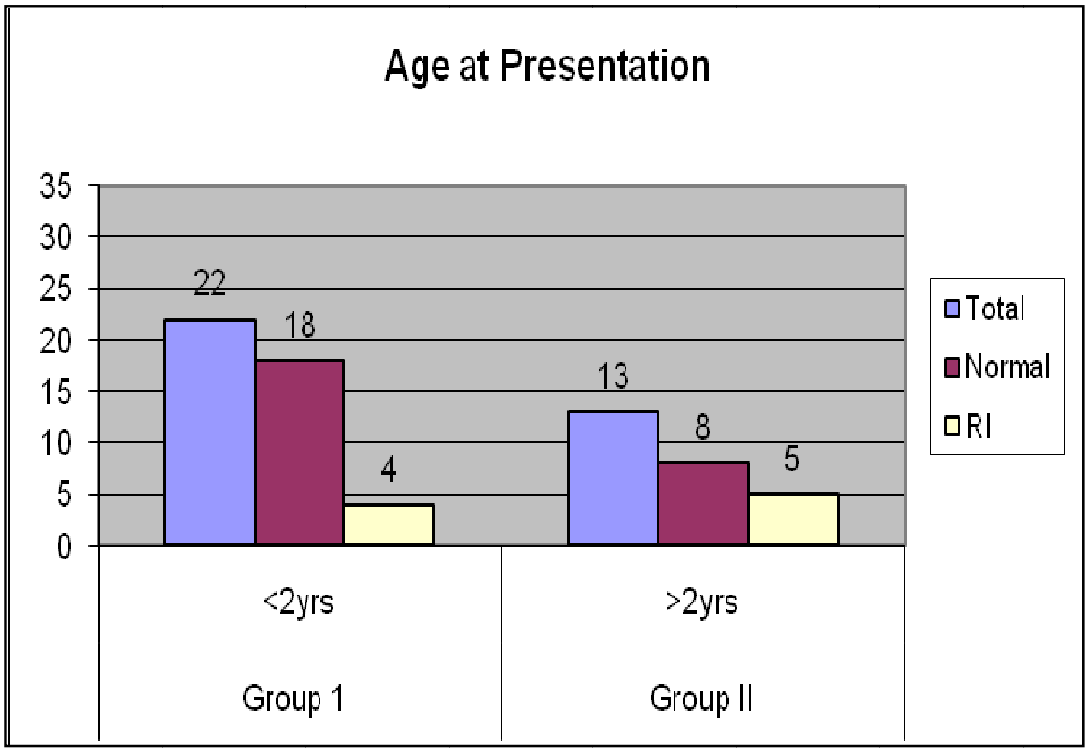
URETEROSTOMY

VESICOSTOMY

OBSERVATION & RESULTS

1.AGE AT PRESENTATION

In group-I, 22 children who presented below the age of 2 years, 81%(18/22) had a normal renal function while just 19%(4/22) progressed to renal insufficiency. Incontrast, of the 13 children who presented above the age of 2 years, 40%(13) of the 5children progressed to renal insufficiency while 60%(8) had normal renal function .

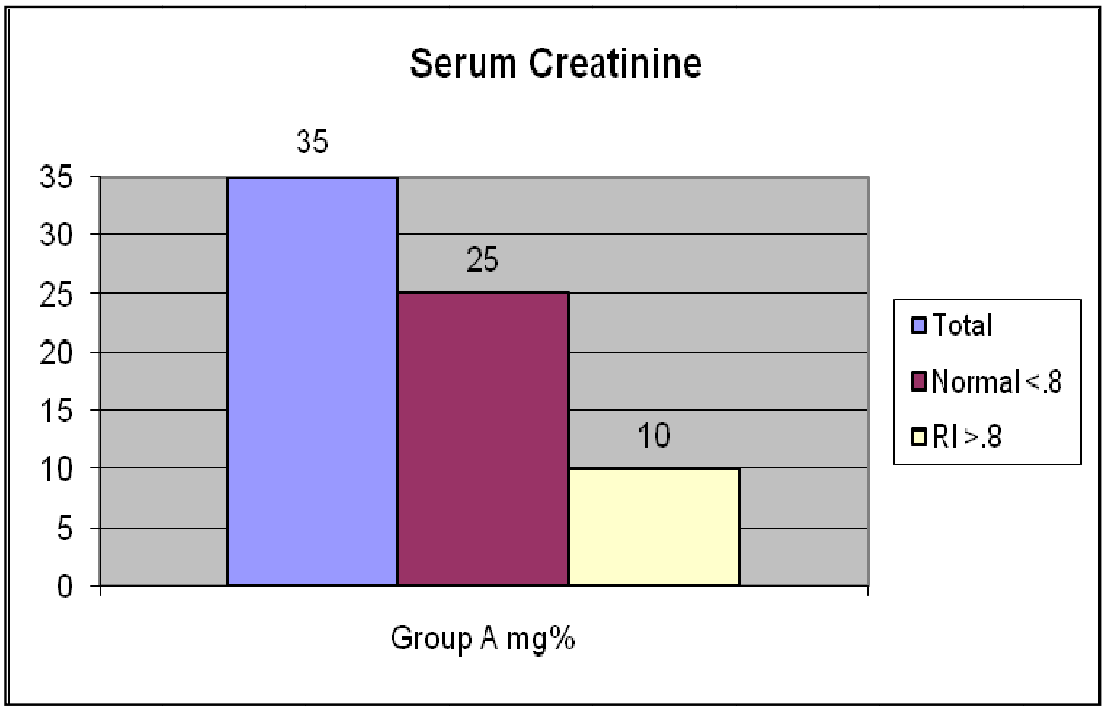


AGE AT PRESENTATION <2YRS

AGE AT PRESENTATION >2 YRS

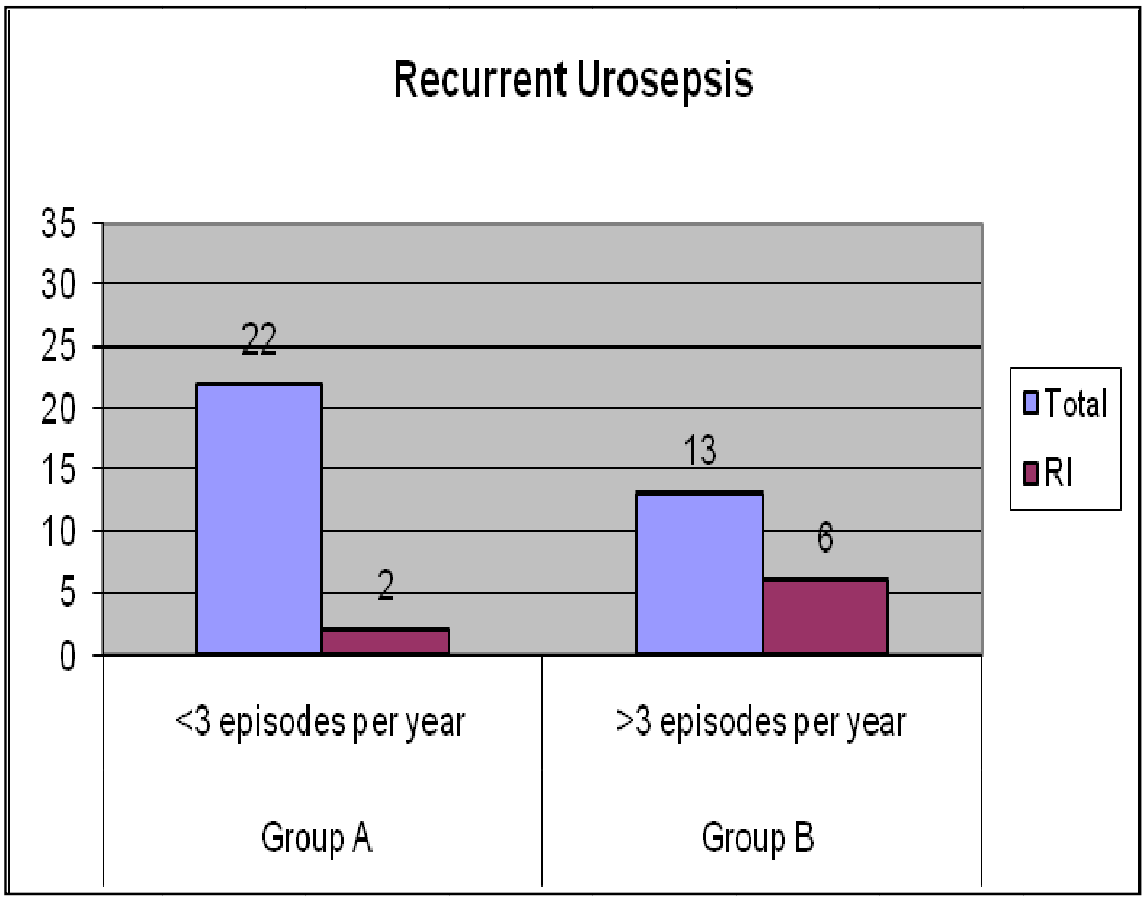
2. SERUM CREATININE LEVEL

Post – valve fulguration , at 3 months follow up in group A 25 patients had a normal renal function, while in group B 10 patients had renal insufficiency, 6/25 of which stabilized to normal renal fuction at 3 years who had initially elevated serum creatinine. Hence the percentage of children with normal renal function at 3 years follow up was 70%, while 30% had renal insufficiency. 10 of these 35 children had a nadir serum creatinine of more than 0.8mg%.



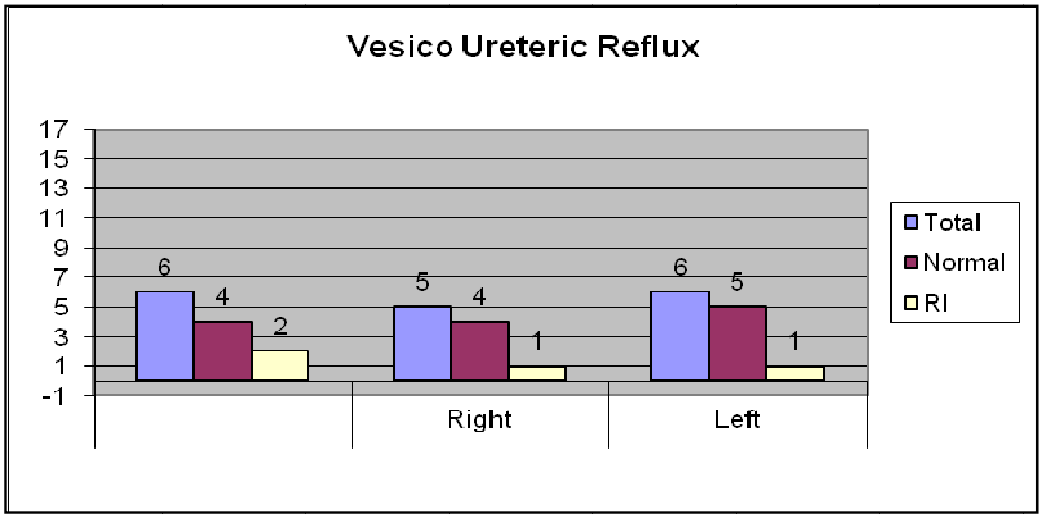
3. UROSEPSIS

13 of the 35 children had evidence of recurrent urosepsis. Poor patient compliance had an important role to play. Of the 13 cases with recurrent urosepsis, 6 progressed to renal insufficiency . Only 2 cases out of 22 childrens with less than 3 episodes or nil urosepsis group progressed to renal insufficiency.



4. VESICO – URETERIC REFLUX

17 of 35 children had vesicoureteric reflux on the initial MCUG.Bilateral reflux were present in 6 children and U/L in children in that right sided reflux noted in 5 cases and left sided 6 cases.10 of which were low grades (I-III) and the remaining high grade (IV – V). 2(40%) of the 6 children with bilateral high grade vesico ureteric reflux had deterioration in renal function at 3 years follow up. There was no effect of surgical treatment(reimplantation procedure) or spontaneous resolution (with chemoprophylaxis) of the reflux on the final outcome. Of the 11 cases of unilateral high grade reflux, 9(80%) had normal renal function , while 2(20%) progressed to renal insufficiency.



NO VUR

UNILATERAL

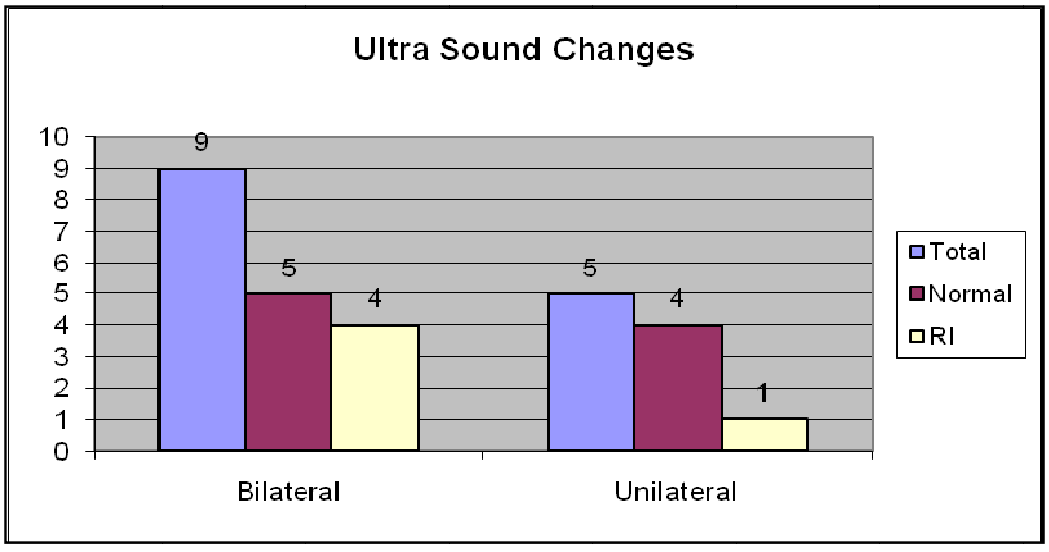
VUR-LT

UNILATERAL VUR –RT

BILATERAL VUR

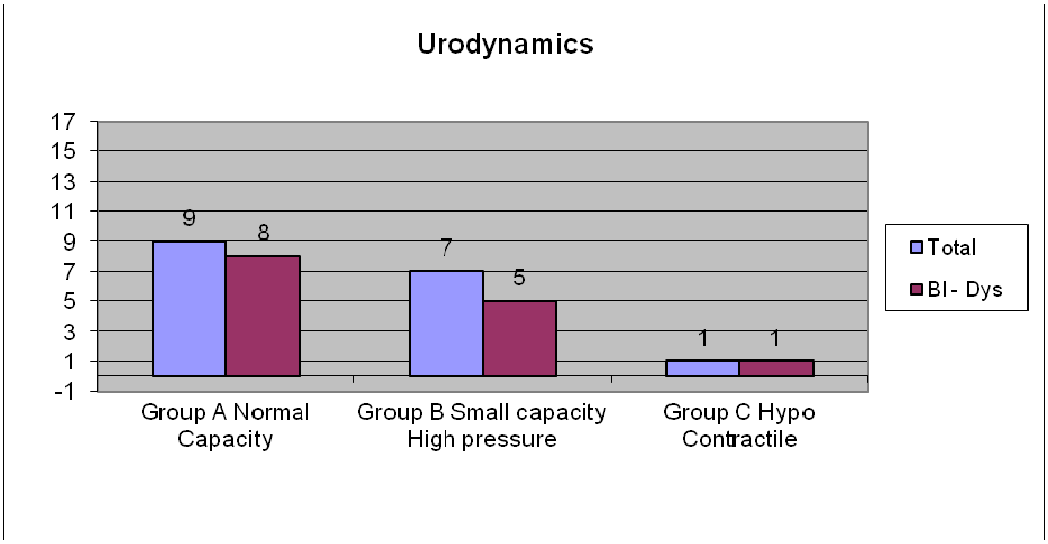
5. ULTRASOUND CHANGES

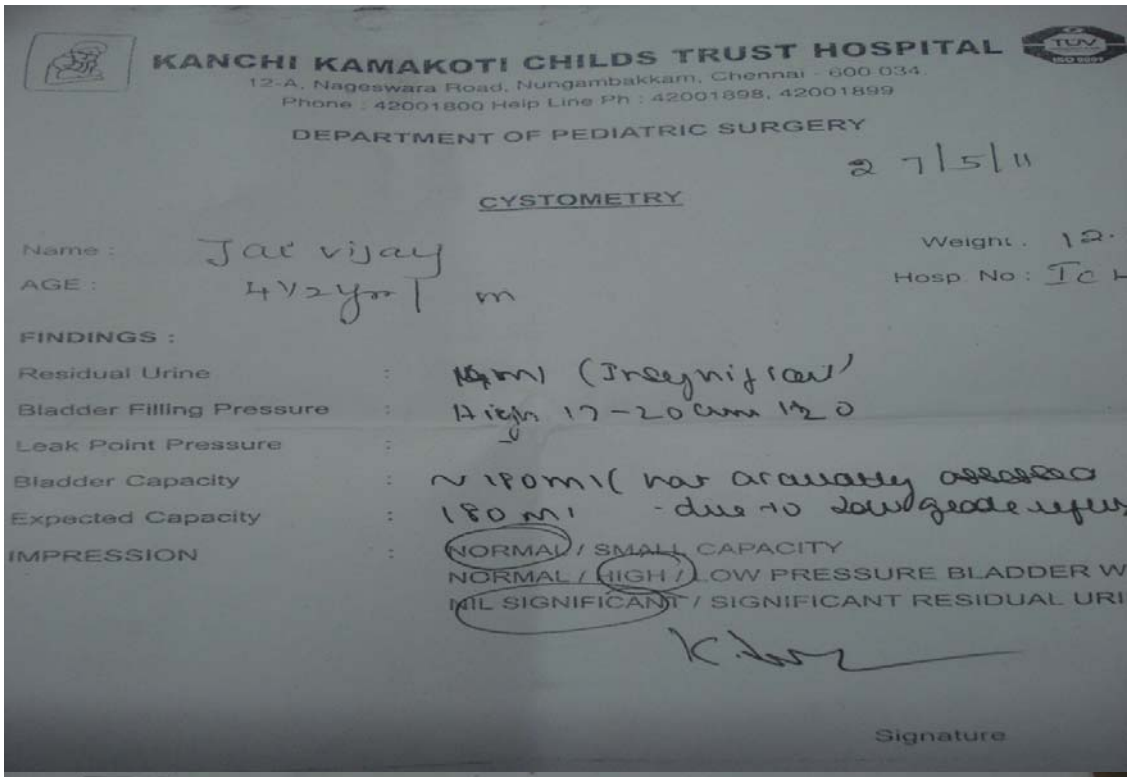
Of the 5 cases with unilateral renal parenchymal damage as seen on ultrasonography, 1 progressed to renal insufficiency while 4 had normal renal parameters. Evidence of bilateral parenchymal damage was seen in 9 cases, 4 of which progressed to renal insufficiency



6. URODYNAMIC STUDY:

Urodynamic evaluation in 17 children with renal insufficiency showed high intravesical pressure with small capacity in 7 cases, high pressure with normal capacity in 9 case and hypocontractile/ detrusor instability bladder in 1 children. Hypocontractile bladders were seen in the older children(age>5 yrs) and were put on clean intermittent self catheterization. Almost 80% of high intravesical pressure, improved with anticholinergic. 2 cases were not on regular medication and those are not improved. 1 child underwent augmentation cystoplasty following failure of anticholinergic therapy. one child died after augmentation due to acute renal failure.





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Phone : 42001800 Help Line Ph : 42001898, 42001899

DEPARTMENT OF PEDIATRIC SURGERY

27/5/11

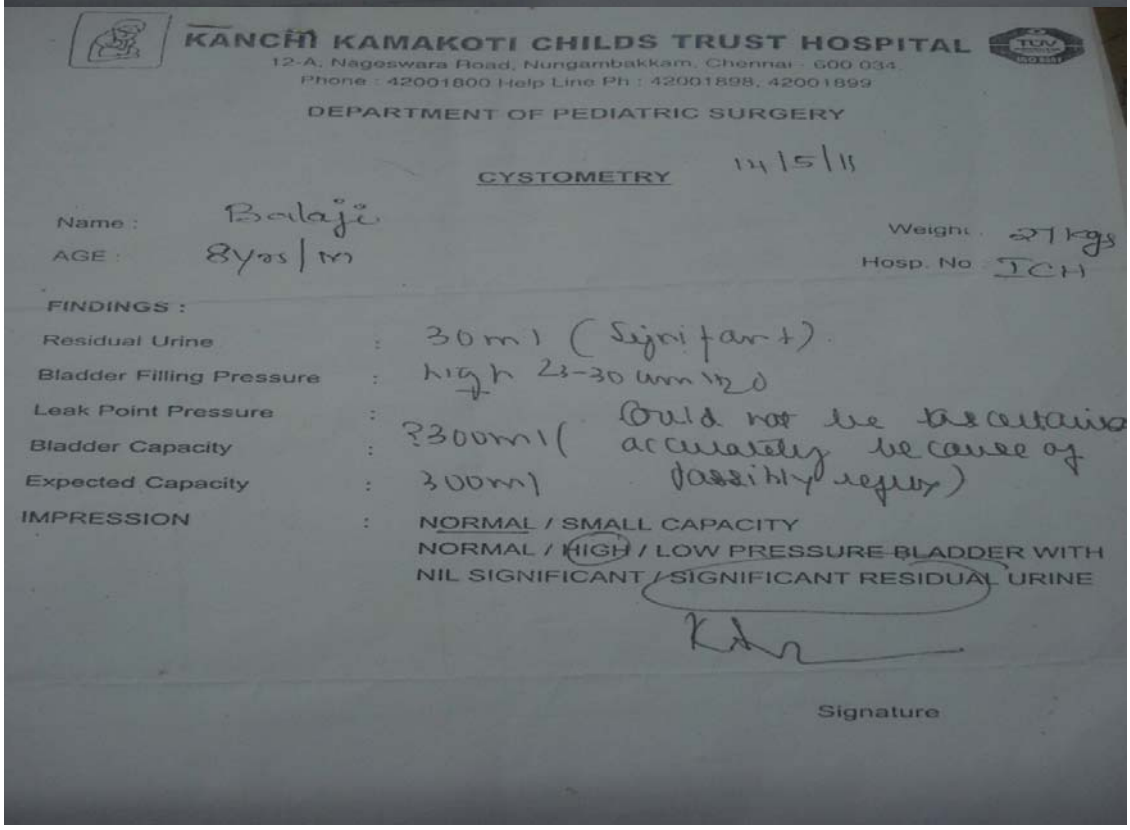
CYSTOMETRY

Name : Jai vijay
AGE : 4½ yrs | m
Weight : 12·
Hosp. No : IC

FINDINGS :

Residual Urine : 40ml (Insignificant)
Bladder Filling Pressure : High 17-20 cm H2O
Leak Point Pressure :
Bladder Capacity : ~ 180ml (not accurately assessed
Expected Capacity : 180 ml - due to vesicoureteric reflux
IMPRESSION : NORMAL / SMALL CAPACITY
NORMAL / HIGH / LOW PRESSURE BLADDER W
NIL SIGNIFICANT / SIGNIFICANT RESIDUAL URI

Signature



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DEPARTMENT OF PEDIATRIC SURGERY

CYSTOMETRY 11/5/11

Name : Balaji
AGE : 8yrs | m
Weight : 27 kgs
Hosp. No : ICH

FINDINGS :

Residual Urine : 30ml (Significant)
Bladder Filling Pressure : High 23-30 cm H2O
Leak Point Pressure :
Bladder Capacity : ?300ml (Could not be accurately because of possibly reflux)
Expected Capacity : 300ml
IMPRESSION : NORMAL / SMALL CAPACITY
NORMAL / HIGH / LOW PRESSURE BLADDER WITH
NIL SIGNIFICANT / SIGNIFICANT RESIDUAL URINE

Signature

DISCUSSION

Posterior urethral valves cause a broad array of renal parenchymal and vesical dysfunction. Because urethral valves are present during the earliest phase of fetal development, primitive tissues mature in an abnormal environment of high intralumial pressure resulting in permanent maldevelopment (hydronephrotic, cystic or dysgenetic kidneys) and long lasting functional abnormalities, with gradual progress towards renal insufficiency. Incidence of renal failure in literature is reported at 15- 40%. Factors possible in defining the final outcome were evaluated in this series.

1. AGE AT INTERVENTION

Our study showed that deterioration of renal function occurred in 19% of children with intervention before age of 2 years, as compared to 40% after 2 years. This finding correlates well with those of Tejani[1] and mayor and associates.

The process of nephrogenesis, which continues to mature till the attainment of maximum glomerular filtration till the age of 2 years, allows some degree of compensation after an early intervention.

In a country like ours, lack of awareness of normal urinary stream and poor patient compliance plays a major role in defining the final outcome . Early referral by primary health centres will facilitate early diagnosis and intervention, which in turn improves renal function.

2.SERUM CREATININE

The baseline serum creatinine after bladder and upper tract drainage indicates the baseline renal parenchymal functional status. 10 out of 35 children with renal insufficiency in this series had sr.creatinine of more than o.8% which was more than twice the normal for their respective age. Hence a serum creatinine of more than 0.8% prognosticates subsequent renal insufficiency.

3.RECURRENT UROSEPSIS

In the current series, the incidence of renal insufficiency in patients with urosepsis was 45%. Recurrent urosepsis >3 episodes in a year (fever with urine culture showing infection) primarily due to poor patient compliance (as regards to follow up and chemoprophylaxis) leads to progressing pyelonephritis and nephron damage and plays an important role in the ultimate outcome of these children.

4.VESICO-URETERIC REFLUX

Vesico-ureteric reflux is present at initial diagnosis in 50% of boys with valves. Bilateral high grade vesico-ureteric reflux is associated with high incidence of renal insufficiency due to associated primary renal dysplasia and recurrent ascending pyelonephritis. In our series 6 children had bilateral high grade vesico-ureteric reflux,2 (40%) of which had renal deterioration.

The role of unilateral reflux as a pop off valve mechanism by buffering the high intravesical pressure, with its protection to opposite kidney, is true only if the contralateral kidney does not show primary dysplatic changes. The incidence of renal insufficiency in cases with unilateral reflux in this series was 20% (2/11). Of these 2 had significant parenchymal damage in the renal unit as seen on ultrasound.

5.RENAL CHANGES ON ULTRASOUND

Ultrasound examination serves to assess the state of renal parenchymal changes as features of renal parencymal damage on ultrasound include increased cortical echogencity, loss of corticomedullary differentiation , and atropic or decreased renal size.

Presence of these factors on ultrasonography hint towards renal insufficiency.of the 5 cases with parenchymal damage as seen on ultrasonography, 1 child progressed to renal insufficiency,while remaing had normal rena parameters. Evidence of bilateral parenchymal damage was seen in 9 cases of which 4

cases progressed to renal insufficiency.hence bilateral renal parencyhaml changes may persist for so many years on ultrasonography but 45% chances of progression towards to renal insufficiency

6.BLADDER DYSFUNCTION

Bladder dysfunction may be associated with posterior urethral valve in 13-38% of patients and may or may not be reversible after relief of obstruction. Urodynamic abnormalties are present in 90% of boys with posterior urethral valves in our series. A urodynamic evaluation should be done in all children above 3yrs after an adequate fulgration or postdiversion closure show presence of urge incontinence, dribbling of urine ,high residual urine, increase in upper tract dilatation or renal insufficiency. In our series, we did UDS for 17 children.

The three major categories of bladder dysfunction:

1.normal capacity, high pressure bladder - 9

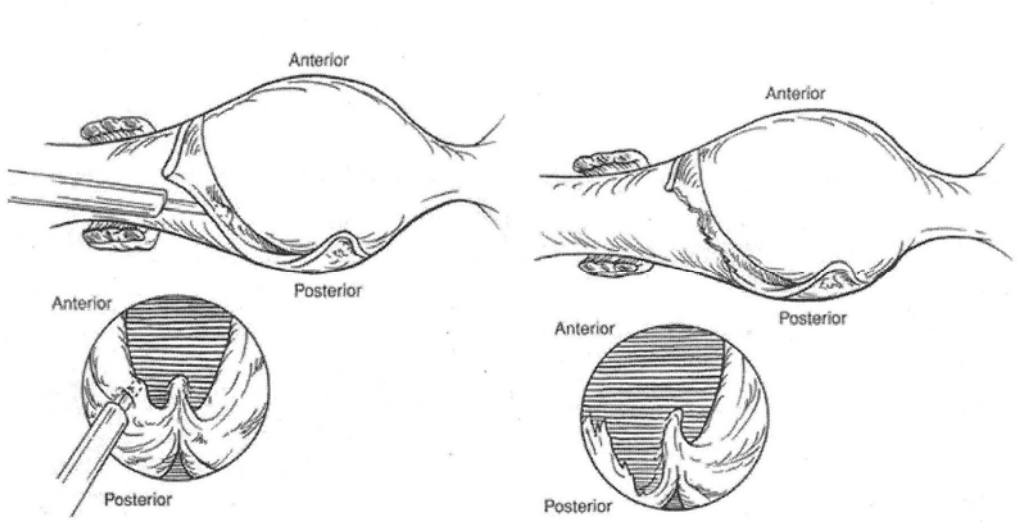
2.small capacity , high pressure bladder - 7

3.detrosor instability (myogenic failure) - 1

These prevent adequate upper tract drainage with subsequent increasing dilatation and damage to upper tracts. Anticholinergic therapy improves bladder compliance, decreases detrusor instability, improves continence and facilitates upper tract drainage in the majority of boys as seen in 9 boys in our series in 6-9months. Clean intermittent catheterization should be done for hypocontractile bladder. Both presented at age of above 5 yrs,indicating the lengthy duration of obstruction leading to decompensation of bladder musculture.

REVIEW OF LITERATURE

ANATOMY AND EMBRYOLOGY

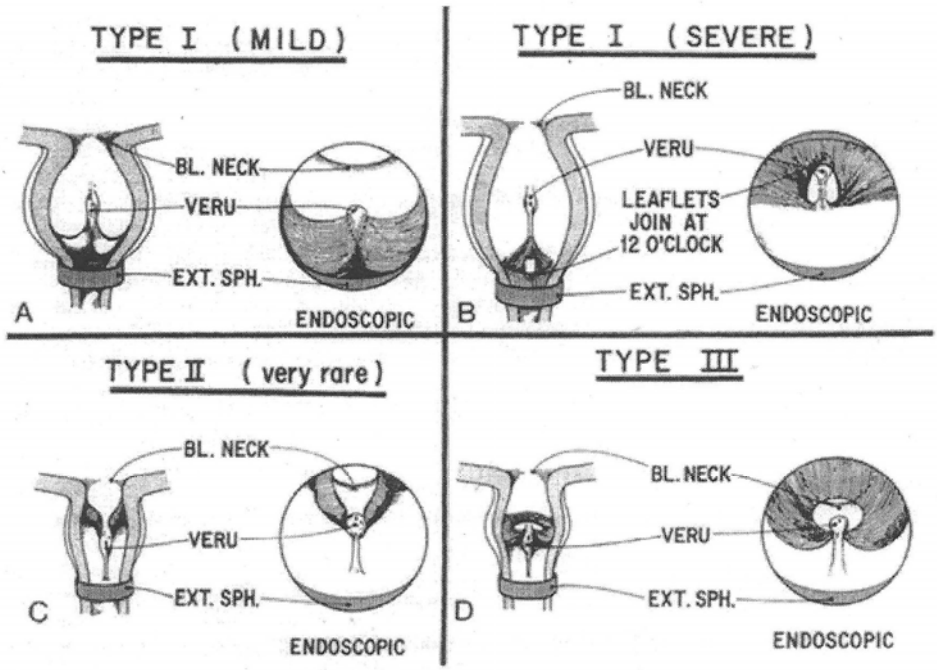


Located in the posterior midportion of prostatic urethra is the verumontanum, containing paired ejaculatory duct openings and urogenital sinus remnant known as prostatic utricle. Extending distally from the veru in the midline is the crista-urethralis and diverging from this are the plicae colliculi,which merge into the membranous urethral area into 2 oblique folds. Tolmatchew in 1870 proposed these to be obstructing in some infants. Stephens postulates that these plicae are normal remnants of the terminal mesonephric ducts which regress during embryogenesis, leaving only the ejaculatory duct openings. Incomplete regression of the plicae leads to fusion dorsally to form the typical PUV. Stephens theory is supported by observations of twins with valves. Livne et al suggests a mesonephric duct origin with possibility of polygenetic

transmission and other undefined factors. BW Young maintains that RUN. obstruction represents abnormal development of urogenital sinus itself (persistence of the sino-vaginal bulb) and not the distal mesonephric duct[4,10,11]

CLASSIFICATION

Dr.H.Hampton Young's long standing system of classification is still employed(1919)



In Type -1 valves are membranes that originate at the verumontanum and travels distally to insert in the anterior proximal membranous urethra with an opening present posteriorly at the verumontanum. The etiology is probably a result of the mesonephric ducts entering the cloaca more anteriorly than normal and fusing in the midline. Such valves offer no

resistance to retrograde instrumentation but coopt during micturition to obstruct urinary flow. It gives characteristic the sail-in-the wind finding in MCU.

In Type-2 folds extend from upper extremity of verumontanum to bladder neck. They are never obstructive and are of historical value only.

In Type-3 valves consist of a ring-like membrane distal to the verumontanum with a perforation present centrally. Field and Stephens describe a variation of type 3 valves in which the membrane becomes stretched so that during micturition it prolapses like a windsock, into bulbar urethra. The cause of these valves is an incomplete dissolution of the urogenital membrane.95% of PUV. are type 1 with variations in leaflet thickness and in the degree of obliquity and coalasence at 12'0 clock position. The other 5% are type 3. Also type 3 is referred as COBB'S COLLAR or MOORMAN'S RING (bulbar urethral obstruction)25. We had 96 type 1 valve patients and 2 type 3 valve patients in our cystoscopy findings

Stephens (1983) suggested **type-4** valves in prune-belly syndrome . Exaggeration of normal folds (plicae) without anterior fusion has been deemed partially obstructive by some (mini-valves). Despite former classifications of PUV, Devan recently stated that most likely there is a single obstructive membrane that may be altered by the passage of urethral catheters or cystoscopes resulting in variable tears of the

membrane. This may be perceived as Type I or III valve. This concept of single type of valve is referred to as congenital obstructive posterior urethral membrane (COPUM)

PATHOPHYSIOLOGY

Above the valves back-pressure effects are nearly always present. These take the form of a widely dilated posterior urethra, a thick walled and usually trabeculated bladder, widely dilated tortuous ureters and bilateral hydronephrosis. V.U.R is common and frequently associated with varying degrees of dysplasia of affected kidney.

The hypertrophy is now understood to be part of the overall detrusor wall thickening that results from the inferior obstruction, and needs no specific therapy, since it is not a true point of obstruction.

Children with P.U.V manifest renal abnormalities that can include varying degrees of dysplasia or hydronephrosis. The renal abnormalities seen in these patients have atleast two theoretical etiologies, pressure and ureteral bud problems.

The first etiology states that the bladder pressure is related to the severity of original obstruction. Dysplasia & /or hydronephrosis develops, based on gestational timing and degree of pressure transmission to the upper tracts, by means of ureteral changes that follow unrelieved bladder pressure elevations.

This occurs with or without breakdown of antireflux mechanism at the U.V junction. It seems likely that this same threshold is accurate in the fetus with valvular obstruction. Even in the absence of reflux, intrapelvic renal pressures reflect the underlying high pressure bladder, when the normal urinary system is first filled to capacity with urine. GFR decreases in a similar setting. The high pressure bladder therefore must play a key role in determining renal compromise but the initial pressure threshold or range that affects renal development in the fetus is yet to be determined.

The second theoretical etiology for renal abnormalities, ureteral bud induction problems, has been examined carefully by Henneberg & Stephens. The severity of renal dysplasia was correlating with the degree of ureteral orifice malposition. They deduced that the abnormality of the ureteral bud & metanephros interaction is causative.

VALVE UNILATERAL REFLUX, DYSPLASIA (VURD SYNDROME)

Ducket et al [2]have emphasized this syndrome of renal dysplasia associated with unilateral reflux. Spurious function on IVP or renal scan with delayed films may be misleading due to reflux. When recognized one can avoid re-implantation of a non functioning system. This syndrome is visualized normally in left, but right also involved some

times. Fortunately this seems to be an effective pressure relieving adaptation which protects the uninvolved side

PRESENTATION

NEW BORN:

More than half of the children with P.U.V are discovered in neonatal period. (P UV= 1: 5000 to 8000 live male births) and 10% are detected prenatally. Presentation in the neonates includes distended bladder, bilateral flank masses, dribbling or weak urinary stream. However a full urinary stream doesn't preclude significant urethral obstruction. Subtle signs of urethral obstruction can include failure to thrive and in some children it may be the only indication of underlying urinary difficulties[3].

An acutely ill baby with P.U.V may have rapid respirations resulting from acidosis. Neonatal urinary ascites or urinary extravasation from kidney is usually considered to be a pressure related phenomenon. At the other end of the spectrum are those fetuses with oligohydramnios, Potter facies and such severe pulmonary hypoplasia that even the most intense supportive measures cannot sustain them. Despite the fact that the obstruction is the primary problem, these infants may not be labeled as P.U.V patients because of severity of their pulmonary compromise and very early mortality.

INFANTS:

Infants in whom the diagnosis has been missed usually present with urinary infection and acute on chronic renal failure. This is generally accompanied by hyperkalemia and severe metabolic acidosis which may lead to respiratory arrest. Water and sodium balance are often also profoundly disturbed. Septicaernia is common and may be complicated by consumptive coagulopathy.

OLDER CHILD:

Older boys may also present with urinary infection but often the main complaint is of a poor urinary stream with straining or dribbling of urine,urinary incontinence. It is important to maintain a high degree of suspicion until urinary abnormalities are excluded in such a child[4]. The less severely affected child may be overlooked in the neonatal period.

DIAGNOSIS & PROGNOSTIC SIGNS

Antenatal Prognostic Factors:

With the advent of maternal antenatal ultrasonography, a significant number of PUV patients are being diagnosed prenatally. Any male fetus with bilateral hydroureteronephrosis and a dilated bladder should be considered to have PUV until proven otherwise. Posterior urethral valves constitute about 10% of all prenatally detected uropathies.

The antenatally detected poor prognostic factors in PUV are :

1. Gestational age at diagnosis 24 weeks.
2. Early onset oligohydramnios
3. Deranged fetal urinary biochemical parameters
4. Incerasd renal cortical echogenicity and cortical cysts.
5. Increased 2 microglobulin in fetal urine.

It was believed that antenatal diagnosis of PUV will improve the outcome by allowing earlier referral and treatment. On the other hand antenatal diagnoosis woule also imply a more severe disease.

Dinneen and co-authors[5] noted a better rental outcome in PUV patients diagnosed antenatally as compared to those who presented acutely within the first 6 months of life. However, subsequent studies do not indicate that antenatal diagnosis improves the outcome in PUV patients.Jee et al reviewed 48 boys with PUV, 17 of which were diagnosed by fetal ultrasonography and 31 postnatally. Renal function on follow-up assessed by serum creatinine was imparied in 59% of those presenting prenatally and in 42% who presented before 3 months of age. Follow-up ranged between 6-90 months in the anitenatal group and between 4-96 months in the postal group. Maternal oilgohydramnios was an important prognostic factor in the outcome. Five of the 6 prenatally diagnosed cases with oilgohydramnis had renal impairment as did one of the two postnatally detected PUV patients who had associated

oilgohydraminos. In this study renal function did not consistently correlate with vesicoureteric reflux.

Antenatally sonography and fetal MRI are useful in diagnosis. In sonography'the typical keyhole sign of bladder and urethral dilatation is seen

In the newborn also, standard imaging studies usually provides an unequivocal diagnosis of posterior urethral obstruction. Sonography again is the preferred initial study when obstruction is suspected, clinically, and will show the typical hydroureteronephrosis, thick walled bladder and wide posterior urethra,cortico-medullary differentiation, associated with PUV.

A loculated urinoma or urinary ascites can also be identified. The MCU usually confirms the diagnosis and demonstrates the reflux present in half of the patients. The dilated posterior urethra, hypertrophied bladder neck, reflux & loss of bladder architecture are typical features.

In doubtful cases diagnosis can be confirmed by cysto-urethroscopy. The valve is best observed with the bladder filled and the endoscope placed well distal to the veru with the water source removed and the connection open. This allows irrigant to flow from the bladder through the instrument, bellowing the cusps demonstrating their coopting margins.

Functional assessment of the kidneys is best obtained with radionuclide renography, allowing quantitation of renal function and cortical scarring. The extraction factor is a simple calculation from the renogram, that reflects the GFR of the individual kidney, based on 1/o uptake of radionuclide in each kidney during the 2-3 minutes of study. Bilateral VUR has usually been associated with both increased morbidity and decreased renal function.

Measure of nadir serum creatinine in the first few months after treatment has also proven to be a useful sign. If the lowest post treatment creatInine is <0.8mg/dI then renal function (with upto 8 years Pf follow-up) has remained in a normal range.

Ducket et al have recently shown that there is a subset of PUV patients that has a good renal function prognosis, due to a **"POP-OFF" mechanism (Large bladder diverticullum, VURD syndrome, urinary ascites, patent urachus). Infants with VURD syndrome (valves, unilateral reflux and dysplasia in a non-functioning unit)** seems to spare the function of contralateral renal unit by a pressure "pop-off" mechanism which apparently protect the nephrons not subject to reflux. This group of patients has significantly better renal function as measured by serum creatinine.

PROGNOSTIC VARIABLES – ANTENATAL [6]

Variable	Good predictors	Poor predictors
In - utero, presentation (weeks)	> 24	<24
Amniotic fluid volume	Normal to moderately increased	Moderate to severly decreased
Sonographic appearance of renal parenchyma	Normal to slightly increased echogenicity	Increased echogenicity to frankly cystic
etal urinary values Sodium (mEq/L) Chloride (mEq/L) Osmolality (mOsm) Urinary output (ml/Hr) Beta 2 microglobulin	< 100 <90 <210 >2 <6	>100 >90 >210 <2 >6

PROGNOSTIC VARIABLES - AFTER BIRTH

Variable	Good predictors	Poor predictors
Sonography - identification of CMJ differentiation	-Present -Pyramids in atleast one kidney	-Absent -Hyperechoic, no pyramids
S. Creatinine	< 0.8 at one year	>0.8 at one year
Reflux	No reflux	Bilateral reflux
Continence	At 5 years	incontinence
Pop off mechanisms Urinary ascites Bladder diverticulum VURD Patent urachus	Present Present Present Present	Absent Absent Absent Absent

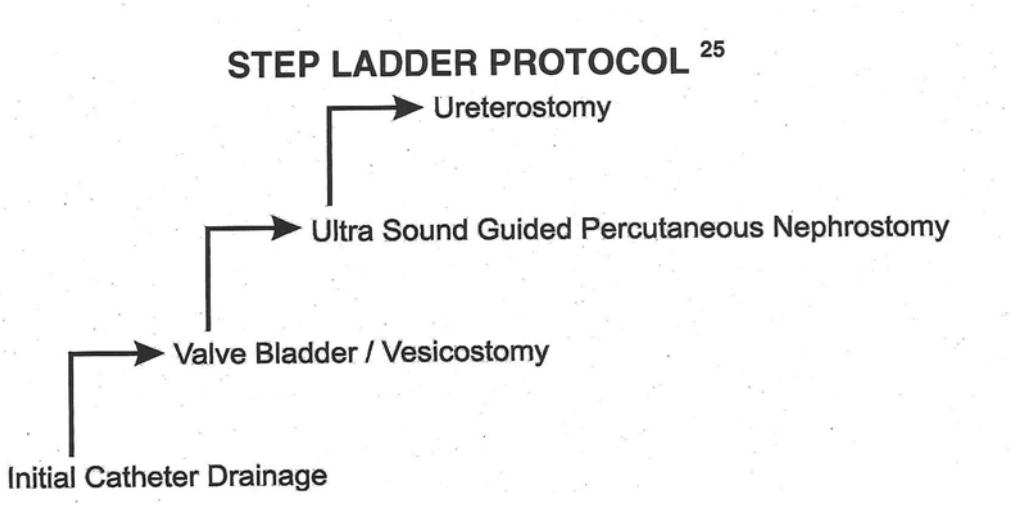
INDIAN SCENARIO OF PUV

The incidence of antenatal diagnosis is only 10% in India. Urinary diversion is done in 50% of cases in our center and 50% of cases are ablated primarily .Only a few cases done during neonatal period. The results of laser fulguration of valves from AIIMS, New Delhi 18 and the primary newborn fulguration from other centers in India are encouraging. Abraham from Kerala, Gopal from Varanasi and Kulasekar from Colombo have developed hooks for vale ablation but the use is limited to their own centers . Fetal surgery is not done in any of our tertiary centres.

As a routine, blood and urine culture are taken. . MCU in newborn is a major procedure and done under strict aseptic precautions. MCU is skipped only in special situations where all information is obtained with ultra sound or radio isotope or rarely when baby is very sick and needs emergency diversion as a life saving procedure. MCU is done later when the baby's condition is stable.

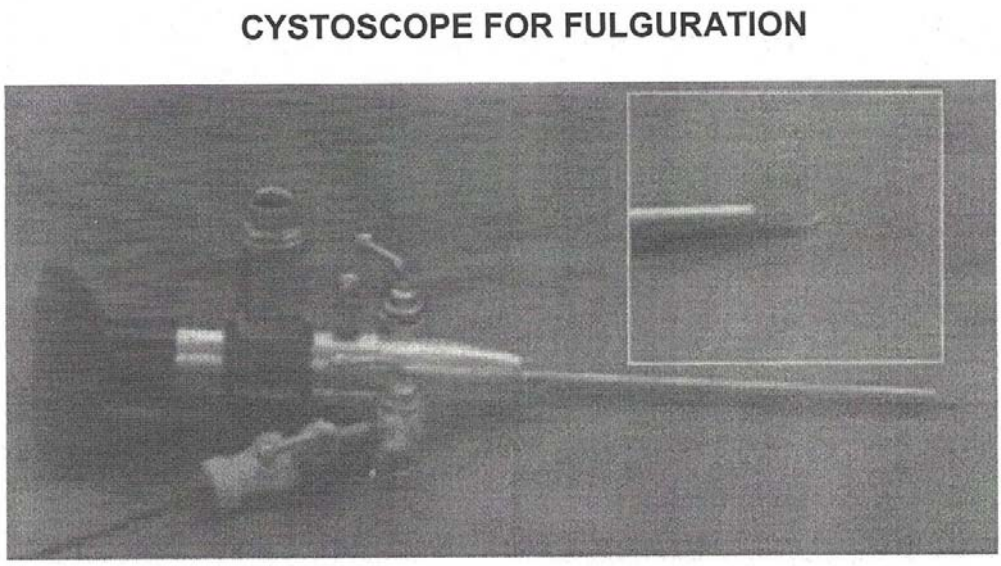
MANAGEMENT PLAN

Hendren et al[7] performed total urinary tract re-construction in newborn period for patients with severe obstructive uropathy due to PUV and included ureteral tapering and re implantation. Primary total re, construction is generally abandoned nowadays because of high risk of obstruction associated with ureteral re impanation into thick and obstructed bladders.



TECHNIQUE OF SURGICAL PROCEDURE –

VALVE FULGURATION



Resection of valves is undertaken using a 6 fr cystoscope fitted with bugbee electrode in newborns with the 3 F working channel[4,11]. For older children 10 fr cystoscope is used. s introduced through the 3F ureteric stent so that only the tip is exposed. Once in the bladder the shape and

position of ureteric orifices if possible are noted and presence of any periureteric diverticulum is noted.

There is no need to excise the valves, but it is necessary to divide the valves at 2 points so that valvular obstruction is destroyed and urinary obstruction is removed. We divide the valves at 2 points 5 and 7'0 clock position. In a rare instance of a diaphragm, we also divide the valve interiorly at two more sides 10 and 12'0 clock positions. Attempts to excise the valve leads to increased incidence of stricture formation. Any remaining free floating tags do not require treatment. It is better to under cut rather than over cut..

If urethra is too small for cystoscope some centers use vesicostomy / ureterostomy to divide the valves.

URINARY DIVERSION –

VESICOSTOMY AND URETEROSTOMY

In uraemic and infected children we do BLOCKSOM VESICOSTOMY is fashioned through a small transverse incision half way between the umbilicus and pubis. The fascia is incised and the peritoneum is pushed superiorly off the dome of the bladder. The urachal remnant is divided and the dome of the bladder is pulled up to the skin. The fascia is secured to the bladder wall to form the required defect (No 22 Fr) and the bladder is matured as flush stoma. We did vesicostomy primarily in 1o: patients.

We did URETEROSTOMY primarily in 8 of our patients . With infant in lateral decubitus position, a postero lateral oblique skin incision is made in the lower flank region. Muscle layer is divided and retroperitoneurn is entered. Dilated tortuous ureter is identified and mobilized enough to reach the skin without tension and divided completely. An infant feeding tube is passed proximally and distally to ensure that there are no angulations. Then ureteral adventia is sutured to the external oblique fascia anteriorly and posteriorly. Muscle and fascial layers are re approximated on both sides of exteriorized ureter.

Trimethoprim sulphamethoxazole is given for months to guard against infection in the healing posterior urethra. This is continued for 9months , in those infants whos cystogram reveals VUR.

FOLLOW - UP

Reflux

Reflux seen in 50% of valve patients. Spontaneous resolution of reflux expected in atleast 20% of these patients after valve ablation. So a period of watchful waiting on prophylactic antibiotics warranted. Reflux has been demonstrated to resolve upto 3 years after treatment.Indications for surgical intervention during this period include break through UTI or massive reflux that interferes with the adequate emptying of system. If re implantation is necessary care must be taken to avoid problems with a thick walled non compliant bladder.

URINARY INCONTINENCE

This is common event when there has been no bladder neck surgery or sphincter damage is seen. Stricture or injury to external sphincter or bladder neck was not taken in our study. Although urge incontinence improves at time of puberty presumably secondary to prostatic growth, this is not always the case. Incontinence may be due to detrusor instability, increased urinary output (Fixed tubular concentrating defect secondary to previous obstruction) or inadequate sensation of a full bladder.

Using formal urodynamic studies 80% of valve population have bladder dysfunction, despite successful valve ablation. Bladder Hypertonia Hyperreflexia and Myogenic failure are the 3 main urodynamic findings and these may be overlapping in individual patients.

Day time urinary incontinence after the age 5 has been a consistent clinical sign in one-third of patients with bad outcome regarding renal function. These findings serve as a warning for early attention to voiding dynamics. Simple relief of obstruction will alter the pathophysiology of the condition in most patients, but sophisticated intervention for the persistent effects caused by secondarily hyper trophied bladder may be necessary. Many of the method now used to control the neurogenic bladder are applicable including anticholinergics,CIC, bladder augmentation with bowel or more recently auto augmentation by creating

a large bladder diverticulum to lower the high bladder pressure and night drainage. In our series one patient underwent augmentation with Mitrofanoff.

Patients with ESRD awaiting transplant must have their bladders evaluated carefully. Failure to address and correct under lying bladder dysfunction could result in ureteral obstruction, reflux, infection or even graft loss.

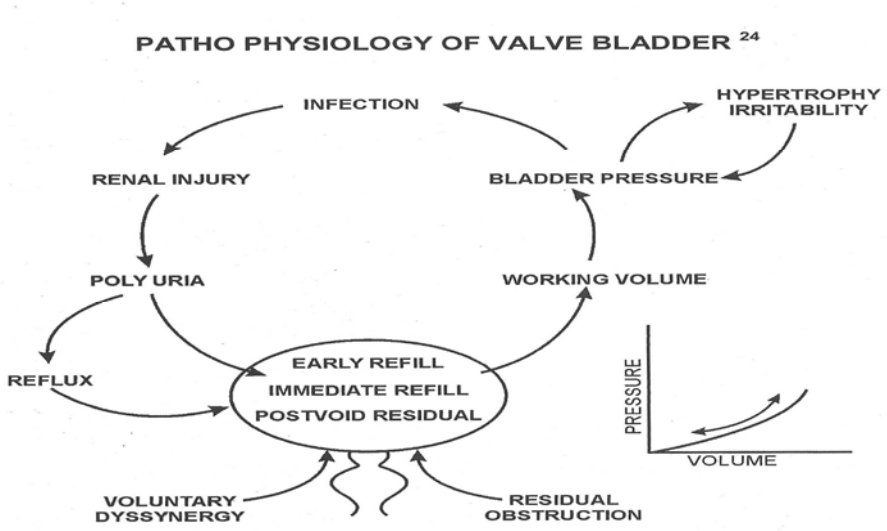
CHRONIC RENAL FAILURE

About 10% of patients with PUV will be stillborn or not survive the neonatal period. Ultimately upto 35% of children with PUV will develop some degree of CRF, generally associated with azotemia, anemia and acidosis. A more precise operational definition is renal insufficiency for more than 3 months with evidence of decreased GFR, anemia, osteodystrophy and electrolyte disturbances. However transplanted valve patient do not do well when compared to transplanted patients with other causes of renal failure. Abnormal bladder dynamics may play a role but clearly more study is warranted. In our study nearly 40 percent of our children progress towardschronic renal failure.

SEXUAL FUNCTION AND FERTILITY

Kruger[8] reported an increased incidence of UDT in 12% of PUV patients. 2 patients had UDT in our study. A recent study indicates that the number of spermatogonia in fetuses with PUV and prunebelly syndrome are decreased when compare to a control group of normal fetuses. Wood house et al reported no evidence of testicular or sexual dysfunction in 20 adults originally treated for PUV. Hence there was a high incidence of retrograde ejaculation and poor ejaculatory force, suggesting persistent bladder neck incompetence. Previously undiagnosed PUV in adults have been reported in infertility patients. These studies emphasis sterility from retained ejaculate in dilated PU and symptoms of chronic prostatitis and bladder neck contracture in others. Adult PUV presentation remains infrequent.

BLADDER DYSFUNCTION – VALVE BLADDER SYNDROME :

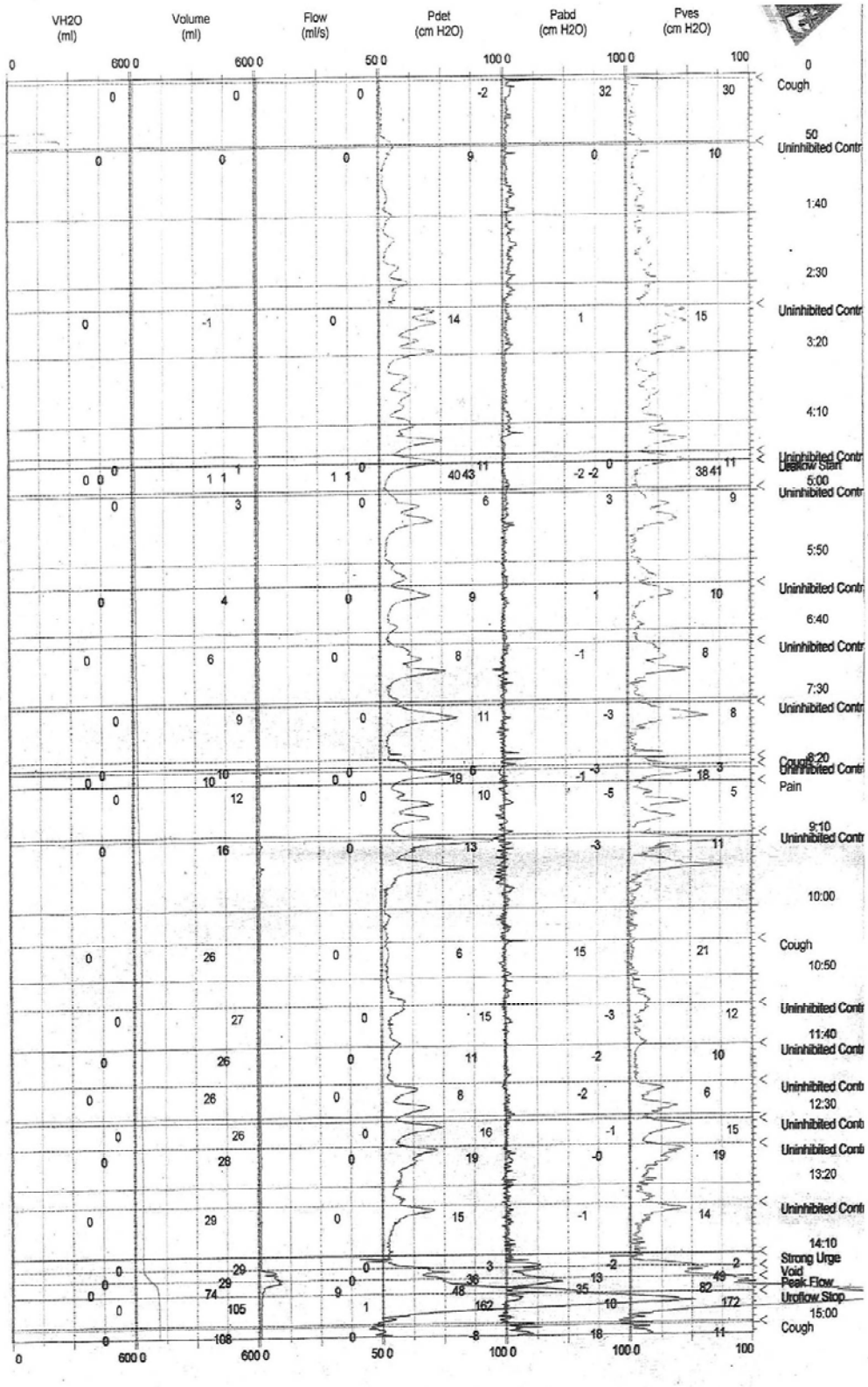


BLADDER DYSFUNCTION – VALVE BLADDER SYNDROME

Even after relief of obstruction, a significant number of patients will continue to have detrusor thickening and poor compliance. Manifestations of this problem include persistent ureteral dilatation associated with full valve bladder syndrome, a physiological obstruction of ureters associated with bladder filling. Even in the absence of reflux, persistent ureteral dilatation secondary to poor compliance of thickened bladder wall can be a source of alarm.

When bladder is empty drainage from the ureters proceeds unimpeded. This abruptly changes with filling of the poorly compliant bladder. Impaired drainage of the upper tracts can be demonstrated with ultra sound., with relief upon voiding or catheterization. This valve bladder syndrome may be managed by complete urinary tract emptying at least twice daily either with double or triple voiding regimen, or CIC along with timed voiding during the day. The use of anticholinergics to reduce the adverse effects of bladder and ureter is also desirable and to prevent upper tract damage. Ureteral changes can be seen in approximately 30% of older valve patients [9]. That is why we had to augment one of our patients (ureterocystoplasty with mitrofanoff) and many are to follow.

URO DYNAMIC STUDY



TREATMENT OPTIONS ACCORDING TO URODYNAMIC FINDINGS:

1. **BLADDER PRESSURE LESS THAN 20 cmH2O**
   ANTICHOLINERGIC DRUGS
   VOLUME EQUAL TO 3-4 HR URINE DRAINAGE
   NIGHT DRAINAGE.

2. **NORMAL CAPACITY & HIGH PRESSURE LEAKAGE**
   ANTLCHOLINERGIC DRUGS

3. **SMALL CAPACITY & HIGH PRESSURE LEAKAGE**
   ANTICHOLINERGIC DRUGS

4. **HYPOCONTRACTILE BLADDER**
   AUGMENTATION + CIC
   (MITROFANOFF)

REVIEW OF LITERATURE:

1.OUTCOME OF CHILDREN WITH POSTERIOR URETHRAL VALVE; PROGNOSTIC FACTOR[10]

This study reviews in 70 children of puv with an aim to identify the prognostic factors & help in defining the end result of implicating the correct treatment & follow-up protocol.

These included age at presentation , intervention less than /more than 2yrs,recurrent urosepsis,vesico-ureteric reflux,renal parenchymal damage as seen on ultrasound, serum creatinine level. 39% had renal insufficiency at the end of 3 yrs follow-up

Factors important in the progression towards renal insufficiency & bladder dysfunction were evaluated. At follow-up-height and weight estimation and urine routine & c/s analysis,sr.creatinine estimation and ultrasound examination were done to assess the progress of the child.MCUG was done at either 3 or 6 monthly follow-up. Indication for a urodynamic evaluation included persistent upper tract dilatation or rising serum

creatinine inspite of an adequate bladder drainage and persistent voiding dysfunction after an adequate valve fulgration.

Factors found to be significant were age at intervention more than 2 yrs,recurrent urospsis, bilateral high grade VUR,renal parenchymal damage on ultrasound & sr.creatinine of more than 0.8 mg%

2.Vesicostomy & delayed fulgration vs primary fulgration for posterior urethral valves . Always a differene in outcome[11]

This study reviews 54 boys of puv with an aim of identify outcome parameters and to both groups.short term outcome parameters included age of fulgration,ultrasound findings, continence status,GFR,& sr. cretinine value.there was no significant outcome differences inthese groups.

CONCLUSION

The incidence of renal insufficiency in children with posterior urethral valves in this series was 38%(30-45%) with an average follow up period of 3 years.

Factors important in prognosticating the progression towards renal insufficiency & bladder dysfunction were:

1. Age at intervention more than 2yrs.
2. Serum creatinine more than .8%.
3. Recurrent urosepsis.>3 episodes/year.
4. Bilaterl high grade reflux.
5. Bilateral parenchymal damage as seen onb ultrasonography.
6. Urodynamics is of immense help in cases having symptoms inspite of good stream .the use of anticholinergics for abnormal urodynamics gives encouragingresults.

PROFORMA

Name: Age :

IP No: XRAY No: U/S No:

Address: Mobile No:

Date of Admission :

Date of 1° Fulguration :

No.of. Fulguration :

Details of Diversion, Fulguration & Closure

Date of Diversion :

Date of Fulguration :

No.of. Fulguration :

Date of Diversion Closure :

Follow – UP Period:

SYMPTOMS

	Pre 1° Fulguration / *Diversion*	Post *Diversion and Fulgurati on*	Post Pre 1° Fulguration / *Diversion Closure*							
			3m	6m	9m	12m	18 m	24m	30m	36m
Fever										
Stream of Urine										
Frequency of Urine										
Color of Urine										
Bed Wetting										
Urge Incontinence										
Failure to thrive										

SIGNS

	Pre 1˙ Fulguration/ *Diversion*	Post *Diversion and Fulguration*	Post Pre 1˙ Fulguration / *Diversion Closure*							
			3m	6m	9m	12m	18 m	24m	30m	36m
1. Temperature										
2. Height										
3. Weight										
4. P/A										
5. Others										

BLOOD INVESTIGATION

	Pre 1˙ Fulguration / *Diversion*	Post *Diversion and Fulguration*	Post Pre 1˙ Fulguration / *Diversion Closure*							
			3m	6m	9m	12m	18m	24m	30m	36m
Sr. Creatinine										
Bld Urea										
K+										
Na+										
Cl-										
HCo3										
Urine R/E										
Urine C/S										

ULTRA SOUND FINDINGS

		RK	LK	RU	LU	UB	TUR
Pre I Fulguration/ *Diversion*							
Post *Diversion and Fulguration*							
	3m						
	6m						
P	9m						
O	12m						
S	18m						
T	24m						
	30m						
	36m						

(RK - Right Kidney, LK- Left Kidney, RU – Right Ureter, LU – Left Ureter, UB – Urinary Bladder, TUR - Turbidity , POST - Post Pre I Fulguration / *Diversion Closure*)

MICTURATING CYSTO URETHROGRAM

		Vesico-Ureteric Reflux				
		Right	Left	AU stream	PU	UB
Pre I Fulguration/ *Diversion*						
Post *Diversion and Fulguration*						
	3m					
	6m					
P	9m					
O	12m					
S	18m					
T	24m					
	30m					
	36m					

(AU – Anterior Urethra, PU – Posterior Urethra, UB – Urinary Bladder , POST - Post Pre I Fulguration / *Diversion Closure*)

OPTIONAL :

URODYNANMICS STUDY	DMSA

INFORMED CONSENT FORM

I ..... father/mother/guardian of .....aged..... Boys was informed by the doctor that my child has posterior uretheral value, for this disease various surgical procedures (primary fulguration / diversion (vesicostomy / ureterostomy) , fulguration & closure) were undergone.

The investigation like blood, Urine samples will be taken in aseptic conditions along with Ultrasoound (KUB) & MCU at free of cost to confirm the diagnosis.

To know the outcome of this disease after definitive procedures, may require extra investigation like DMSA, urodynamic study may be required in selected cases & I will pay the required in sum of money to get the investigations done if needed.

I therefore agree to get my child participate in this study with my own knowledge & I will provide the correct information needed there will be no objection from my side for my child's examination & investigation.

I ..... father / mother / guardian of under mentioned child do hereby agree & allow my son to participate in the study.

I confirm that I have been told about this study in my mother tongue & have had the opportunity to ask question.

I confirm that I have been told about the risk & potential benefits for my child's awards participation in the study.

I understand that my child's / wards participation is voluntary and I have the right to withdraw my child/ward from this study at any part of time without giving any reasons and without my child's/wards benefits being affected.

I agree not to restrict the use of any data or results that my arise from this study.

Name & Address of the parent/guardian .....

Signature/thumbprint of parent/guardian .....

Signature of medical officer .....

Witness signature .....

Date .....

Principal investigator

Address

Date

தகவலளிக்கப்பட்ட ஒப்பந்தல் படிவம்

_____ என்ற ஆண் குழந்தையின் தாய் அல்லது உறவினராகிய _____ என்ற என்னிடம் குழந்தைக்கு Posterior Urethral Valve என்ற நோய்க்கு தக்க அறுவை சிகிச்சை செய்து இருப்பதை மருத்துவரால் தெரிவிக்கப்பட்டது. இதற்கு பிறகு வரக்கூடிய பின் விளைவுகளைப் பற்றியும் தெரிவிக்கப்பட்டது. இதை வராமல் தடுக்க தக்க பரிசோதனை செய்ய குழந்தையின் இரத்தத்தில; Cretinine, Urea, Urine C/s, Ultra Sound, மற்றும் MCUG படமும் எடுத்து பரிசோதனை செய்வது அவசியம் என்று தெரிவிக்கப்படுகிறது.

நோயின் தன்மையை பற்றியும் அதன் பின் விளைவுகள் பற்றியும் மருத்துவரால் தெளிவாக எடுத்துரைக்கப்பட்டது. இந்த ஆய்வில் இரத்தம்/சீறுநீரக பரிசோதனை USG மற்றும் MCUG சுகாதாரமான முறையில் இலவசமாக செய்யப்படும் என்று மருத்துவர் தெரிவித்தார். இந்த நோயினால் சீறுநீரகம் மற்றும் சீறுநீரகபை நிலைமையை அறிய DMSA அல்லது URODYMNAMICS படமும் தேவைப்படலாம் என்றும் தேவை என்றால் அதற்குண்டான உரிய தொகையை செலுத்தி பரிசோதனை செய்து கொள்ள சம்மதிக்கின்றேன்.

இந்த ஆய்வு பற்றி எனக்கு விளக்கமாக எனது தாய்மொழியில் சொல்லப்பட்டது. இந்த ஆய்வில் பங்கெடுத்து கொள்வதால் எனது குழந்தைக்கு ஏற்படக்கூடிய அபாயங்கள் மற்றும் நன்மைகள் பற்றி எனக்கு விளக்கப்பட்டது. எனது குழந்தைக்கு வழக்கமாக செயல்படும் மருத்துவ கவனிப்பு அளிக்கப்படும் என்று தெரிவிக்கப்பட்டது.

இந்த ஆய்வின் எனது குழந்தையை பங்கெடுத்துக் கொள்ள முழுமனதுடன் சம்மதிக்கிறேன். கேள்விகள் கேட்பதற்கு எனக்கு வாய்ப்பளிக்கப்பட்டுள்ளது.

இந்த ஆய்வில் பங்கேற்பது தன்னிச்சையானது, எந்த நேரத்திலும் என் குழந்தை பங்கேற்பதை எந்த விளக்கமும் தராமல் நிறுத்திக் கொள்ளலாம்.

மற்றும் இதனால் என் குழந்தைக்கு கிடைக்க வேண்டிய மருத்துவ சிகிச்சைக்கு எந்த இடையூறும் ஏற்பாது என எனக்கு தெரிவிக்கப்பட்டது.

எனது குழந்தையிடமிருந்து பெறப்பட்ட இரத்த மாதிரிகள் உயர் பரிசோதனை கூடங்களுக்கு அனுப்புவதற்க்கு சம்மதிக்கிறேன்.

இந்த ஆய்விலிருந்து கிடைக்கும் முடிவுகளை பயன்படுத்துதை கட்டுப்படுத்தாமலிருக்க நான் சம்மதிக்கிறேன்.

குழந்தையின் பெற்றோனர்

கண்காணிப்பாளர் கையெழுத்து_____

தேதி:

எழுதப்படிக்கத் தெரியாத பெற்றோர்/கண்காணிப்பாளர்

கைகக்கட்டை விரல் ரேகை.

சாட்சியின் பெயர்	:	.....
சாட்சியின் கையெழுத்து	:	.....
தேதி	:	.....
ஆய்வாளர் பெயர்	:	.....
ஆய்வு வழிநடத்துபவர் பெயர்	:	.....
கையெழுத்து	:	.....
தேதி	:	.....

MASTER CHART:

S.NO	NAME	AGE	IP.NO	Age at presentation	Procerude	Sr.creatinine	Urine H/b	Urine c/s	u/s (parenchymal damage)	MCU Reflux	UDS	DMSA
1	Hari	2	627313	<2yrs	F	<0.8	_	<3	_	-	_	_
2	Sanjay	5	627739	<2yrs	F	<0.8	_	>3	B/L	U/L	NC/HP	_
3	Thiyga	3.3	626466	>2yrs	F	<0.8	_	<3	_	_	_	_
4	Vijay	2.6	626044	<2yrs	VE	<0.8	_	<3	_	_	_	_
5	Mahakrishnan	5	625480	>2yrs	VE	>0.8	+	>3	B/L	B/L	SC/HP	SCAR
6	Partha	2.8	625200	<2yrs	UR	>0.8	+	>3	U/L	U/L	_	SCAR
7	Monish	6	626449	<2yrs	VE	<0.8	_	<3	_	_	SC/HP	_
8	Saimunich	4.9	625200	<2yrs	UR	>0.8	+	>3	U/L	U/L	NH/HP	SCAR
9	Manavala	3	625449	>2yrs	F	<0.8	_	<3	_	_	_	_
10	Naveen	3.5	625258	>2yrs	F	<0.8	_	<3	_	_	_	_
11	Asik	6	625567	>2yrs	VE	<0.8	_	<3	_	_	_	_
12	Suriyaprakash	2.8	625544	<2yrs	F	<O.8	+	>3	B/L	U/L	SC/HP	_

13	Sakthivel	4.6	625533	<2yrs	UR	>0.8	_	>3	U/L	U/L	NH/ HP	SCAR
14	Sujith	5.6	624988	<2yrs	F	<0.8	_	<3	B/L	U/L	_	_
15	Syed sulaiman	3.8	624295	<2yrs	UR	<0.8	_	<3	_	U/L	_	_

16	Balaji	3	623796	>2yrs	F	<0.8	_	<3	_	_	_	_
17	Diwakar	7.2	624265	<2yrs	VE	>0.8	+	>3	B/L	B/L	SC/ HP	SCAR
18	Ajith	3.2	623989	>2yrs	F	<0.8	_	<3	B/L	U/L	_	_
19	Jaffer	6	615071	>2yrs	VE	<0.8	_	<3	_	_	SC/ HP	SCAR
20	Ganesh	3.11	621164	<2yrs	VE	<0.8	_	<3	_	_	_	_
21	Karuna	5.5	620832	>2yrs	F	>0.8	+	>3	_	_	HC	_
22	Lakshman	3	620451	<2yrs	F	<0.8	_	<3	_	_	_	_
23	Dinesh	4.6	618052	<2yrs	VE	<0.8	_	<3	_	_	SC/ HP	SCAR
24	Aravind	1.11	618788	<2yrs	UR	>0.8	+	>3	U/L	U/L	-	_
25	Vasanth	5.8	621006	<2yrs	VE	<0.8	_	-<3	_	_	SC/ HP	SCAR
26	Subash	4.6	618798	<2yrs	F	>0.8	+	>3	U/L	U/L	NC/ HP	_
27	Ranjith	3.2	679909	<2yrs	UR	>0.8	+	>3	B/L	B/L	_	_
28	Madan	3	619885	>2yrs	F	<0.8	_	<3	_	_	_	_
29	Keerthir	8.0	619573	>2yrs	F	<0.8	_	<3	B/L	B/L	NC/ HP	SCAR

30	Roshan	3.1	620622	<2yrs	UR	<0.8	_	>3	_	U/L	_	–
31	Vishwa	3	621231	<2yrs	UR	<0.8	_	<3	_	U/L	NC/ HP	–
32	Arun	7.2	621473	>2yrs	F	<0.8	_	<3	_	_	_	_
33	Vishnu	5.3	628974	<2yrs	VE	>0.8	+	>3	B/L	B/L	SC/ HP	SCAR
34	Vetri	4.9	628013	<2yrs	F	<0.8	_	<3	_	_	_	–
35	Mugesh	6.5	627349	>2yrs	F	<0.8	_	<3	_	_	NC/ HP	–

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F-FULGRATION, VE-VESICOSTOMY,UR-URETEROSTOMY,
URINE R/E-ALBUMIN +,URINE C/S->3 EPISODES /YR
U/S- PARENCHYMAL DAMAGE , B/L-BILATERAL,U/L-UNILATERAL
MCU-MICTURATING CYSTOURETHROGRAM
UDS-URODYNAMIC STUDY; NC/HP-NORMAL CAPACITY/HIGH PRESSURE,SC/HP-SMALLCAPACITY/HIGH PRESSURE,HC-HYPOCONTRACTILE
DMSA SCAN

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